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THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

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HONORARY VETERINARY SURGEON TO HIS MAJESTY THE KING,
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CAVALIERE DEI SS. MAURIZIO E LAZZARO (ITALY),
HONORARY MEMBER OF THE AMERICAN VETERINARY ASSOCIATION,
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CONTENTS

FRONTISPIECE AND OBITUARY— PAGE

The late Captain G. H. Bennet, M.R.C.V.S. ... ... 54 & 55

EDITORIAL—

Foot-and-Mouth Disease and the Veterinary Officials of the Ministry of Agriculture 56

GENERAL ARTICLES—

Graves' Disease and Thyroid Instability on the Cow, and its Relation to Ovarian Disease. By L. P. PUGH, B.Sc.Lond., F.R.C.V.S. 58

Some Common Causes of Poisoning among Domestic Animals. By G. W. CLOUGH, D.Sc. 70

An Outbreak of Chicken Cholera. By A. KNYVETT GORDON, M.B. ... 73

CLINICAL ARTICLES—

Poisoning from Castor Beans. By Maj.-Gen. Sir JOHN MOORE, K.C.M.G., C.B., F.R.C.V.S. 75

Acute Lead Poisoning in a Mare. By R. GREGG, M.R.C.V.S. 76

An Unusually Large Elbow Tumour. By J. D. FULTON, M.R.C.V.S. ... 78

Cake Poisoning. By R. HUDSON, F.R.C.V.S. 79

Hydrocyanic Acid Poisoning in Young Calves. By P. WILSON, M.R.C.V.S.... 79

Notes *re* Lead Poisoning. By E. P. EDWARDS, M.R.C.V.S. 81

An Unusual Case of Lead Poisoning in Cattle. By HARRY P. STANDLEY, M.R.C.V.S 87

Yew Tree Poisoning. By HUGH BEGG, F.R.C.V.S. 88

Rhododendron Poisoning in Cattle. By WM. LOTHIAN, M.R.C.V.S. ... 88

Castor Oil Seed Poisoning in Cattle. By PHILIP VINCENT, M.R.C.V.S. ... 89

Bracken Poisoning in Pigs. By JAMES BLAKEWAY, M.R.C.V.S. 89

Toxicology Notes. By G. MAYALL, M.R.C.V.S. 90

REVIEWS 91

NOTICES... 92

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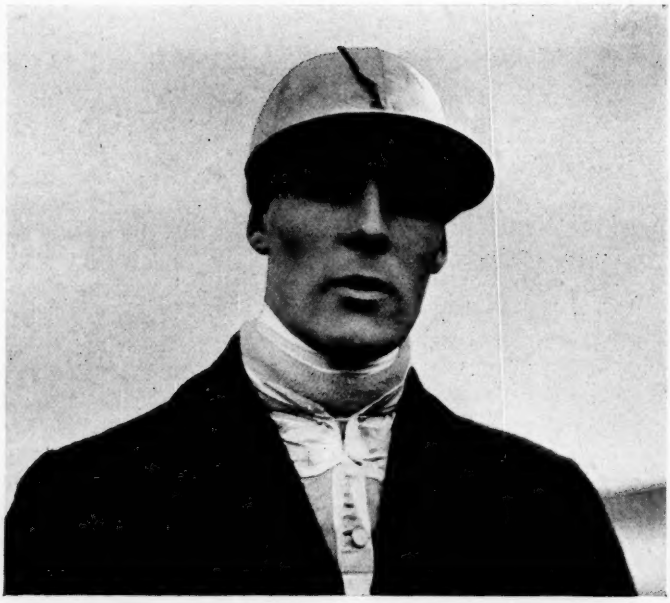

THE LATE CAPTAIN G. H. BENNET, M.R.C.V.S.

Winner of The Grand National on "Sergeant Murphy," 1923.

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FEBRUARY, 1924.

Obituary

THE LATE CAPTAIN GEOFFREY HARBORD BENNET, M.R.C.V.S.

THE profession will learn with regret of the fatal sequel which has terminated the accident to Captain G. H. Bennet, M.R.C.V.S., of Newmarket. As a gentleman rider he had attained a fame in the sporting world which has fallen to the lot of very few, and he was as well known for his clean riding as for his successful horsemanship. Born near Bury St. Edmund's, he qualified in 1916, when only twenty-two years of age, from the London College and immediately took his part in the Great War. He served both in France and in Egypt, winning many races for the unit to which he was attached. He was deservedly popular with all classes, and his popularity has followed him throughout the whole of his career. After the War he settled down at Newmarket, where he took up steeplechase riding and soon became one of the most prominent amateur jockeys. On three occasions he rode in The Grand National, and it was his clever horsemanship which brought him the first place last year on "Sergeant Murphy." He was for a short time in partnership at Newmarket with the late Mr. John Coleman, M.R.C.V.S., but his racing career left him little time for ordinary practice.

As one of our graduates who was a good sportsman Captain Bennet "made good" for the profession, and we can ill afford to lose him, particularly at the commencement of a career which appeared to give such brilliant promise. Had he lived, there is no doubt that he would have achieved the highest possible place in the list of English gentlemen riders.

XUM

Editorial

FOOT-AND-MOUTH DISEASE AND THE VETERINARY OFFICIALS OF THE MINISTRY OF AGRICULTURE.

THE veterinary officials of the Ministry of Agriculture are passing through a thankless and tedious time at the present moment, and the amount of abuse which they have been receiving in the Public Press (in many instances, unfortunately, from those who ought to have known better) has been enough to make every hair on their respective heads turn grey. Amongst those who would without hesitation take in hand the foot-and-mouth question (if allowed) and cure at least 100 per cent.! are to be noted old ladies, clergymen, fossils of various kinds, and even members of the medical profession; in fact, everybody seems to know the Veterinary Surgeons' job better than the Veterinary Surgeon himself, and the astonishing part is, that the editors of newspapers can be gulled into printing such twaddle as has recently appeared. Even sensible journals have been drawn into the net, and the man in the street can readily be excused if he believes that the members of the veterinary profession are uneducated, unscientific and untrained. For one thing, however, on this occasion, we are to be congratulated, and that is, that at all events we have been loyal to ourselves and in our support of the veterinary authorities at the Ministry of Agriculture, for the response which has been given to the appeal for temporary help in this national peace crisis has been as spontaneous (and as great in proportion) as was the help given voluntarily in response to the appeal of the Director-General of the Royal Army Veterinary Corps during the time of war. It is good for our heads of departments to be able to realise that in a national crisis, whether it be one of peace or of war, the members of the profession have ever been ready to jump into the gap; and when this present outbreak has been stamped out (as it surely will be) it will be pleasant for them to look back on the loyal help by which it was done. The stamping-out process may appear to the unreasoning general public to be crude and cruel, but in reality it is nothing of the kind. It is in more ways than one the means of doing the greatest good to the greatest number, and it is appalling to think what would have happened to the whole of the country had this vigorous policy not been sternly adhered to. There is no question but that very many counties of Great Britain, which are now entirely free, would have had the foe in their midst, and it is not as if we had a disease to deal with which gave immunity after one attack. Once endemic in the country, it would be a matter of millions per annum before it could be got rid of; and the stamping-out process would have to be resorted to at the finish.

A further proof of the loyalty of the profession to those of its members who are in authority at the Ministry, was demonstrated by a resolution which was unanimously passed at a well-attended meeting of the Central Branch of the National Veterinary Medical Association held at 10 Red Lion Square, on Thursday, January 3rd, in which it was announced: "That this meeting of the Central Branch of the National Veterinary Association express the utmost confidence in the veterinary officials of the Ministry of Agriculture in the measures they consider necessary to adopt in their present difficult position in having to deal with so severe an outbreak of foot-and-mouth disease."

It was felt, and expressed, that the veterinary officials of the Ministry had a better grip and understanding of the method of dealing with this disease than anybody else in the country; and it was thought, by wording the resolution in this way, that it would be some little encouragement to them to stick to their guns, and to follow up whatever opinion they deemed to be best, whether complete slaughter, or a policy of isolation in certain well-considered instances. It was a counterblast to the rabid and hysterical opinions which have been brought forward at some of the meetings in the badly affected districts. This has been followed up by similar resolutions before the National Veterinary Council and several other Veterinary Societies; all passed unanimously. The sympathies of everybody are with those who have been the victims and the losers, but that is no reason why the most stringent and stern efforts should not be made to confine it to the present infected areas and prevent its extension to the whole of the country, with its sequel of additional victims and more losers.

The agricultural community, and, indeed, too, the general British public, ought to be grateful for the fact that it has a well organized Veterinary Department, which although poorly paid and understaffed, has loyally tackled one of the most difficult fights against disease which it has ever been necessary to encounter. A quotation from an article in an American contemporary (*Veterinary Medicine*) for January 1923, on "The Veterinary Situation in America," by Dr. Tarkas of Chicago, says:—

"Among the contagious diseases, foot-and-mouth disease is the greatest menace, not only because the animals cannot be worked and the cows do not give milk during the period of illness and quarantine, but also because most of the calves and some of the adults perish; and besides, the same individuals in many cases suffer from the disease twice in a single year.

"In spite of the efforts of the veterinarians the disease could not be controlled on account of the fact that the people did not take quarantine seriously (violation of the laws by dealers who peddled animals

from infected yards, etc.) and the disinfections in many instances were not properly done. Discouraged with this attitude of the people, the veterinarians for a time paid no attention to the official report coming from village officials, but finally owing to the seriousness of the situation the people themselves who had before shown antipathy towards sanitary regulations began to appeal directly to the veterinarians for strict quarantine orders and to personally control exact disinfection. Quite naturally, having from thirty to sixty villages to attend to, the veterinarians were unable to supervise the entire work."

And it does not require a very big stretch of imagination to apply this as a " might have been " to our own country.

General Articles

GRAVES' DISEASE AND THYROID INSTABILITY IN THE COW, AND ITS RELATION TO OVARIAN DISEASE.*

By L. P. PUGH, B.Sc., Lond., F.R.C.V.S.,
Sevenoaks.

Introduction.

Observations extending over some years have at length led the writer to the assumption that the so-called " nymphomania " of cows is merely one phase of a process which is in its entirety analogous to the hyperthyroidism-myxœdema syndrome in the human species. But to establish this thesis is a matter of no small difficulty. The symptom most readily discovered in cows, i.e. the sexual excitement, may be carefully concealed in human beings. On the other hand, the vascular changes which can be readily studied in man are not easy and often impossible to elucidate in animals. These very facts make the task more urgent, since here is an obvious example (if the thesis be proved) of the great mutual assistance which veterinary and human pathology may afford each other. Study of this subject has naturally opened up more lines of inquiry than the mere identification of the two diseases, but in this paper it is proposed to advance and support, however inadequately, the following propositions, namely :—

(1) That " nymphomania " in cows is a part of a constitutional disorder known in human pathology as Graves' disease.

(2) That disorder of the thyroid function is an essential feature of the " nymphomania " syndrome.

(3) That this disorder is profoundly influenced by ovarian disease.

* *Proceedings of the Royal Society of Medicine*, July 1923. A preliminary note was published in the *Veterinary Journal*, vol. xxix, 1922, p. 382.

It is impossible in cows, and, as the writer imagines, difficult in human beings, to separate with certainty the symptoms of hyperthyroidism from those of myxœdema. It is true that at one time physicians saw Graves' disease as one well-defined syndrome and myxœdema as another. Later, these were found to be much more closely connected than had hitherto been considered probable. Later still the conceptions of hyper- and hypo-thyroidism have extended into an ever-widening sphere until the original clear-cut syndromes have begun to be obscured by the results of clinical and experimental observation.

Thus it comes about that nymphomania in cows cannot be presented as a syndrome due to the disorder of a single endocrine gland, but rather as a disturbance of that endocrine balance which we call

FIG. 1.
Cow in good health.

good health, a balance to which each organ contributes its proper share of some specific secretion. There are indeed many such disturbances, nor can we say with certainty which gland is primarily at fault in each of them. But from many which are known we pick out Graves' disease in man and nymphomania in cattle because they are seen to have so many features in common that their comparison seems likely to be the most fruitful line of research in approaching the many problems which await solution in endocrinology and in practical therapeutics.

When we speak of Graves' disease that name must be considered to apply to the whole symptom-complex of which a portion was first described by Parry in 1825, then by Graves in 1835, later by Basedow in 1840, while a further portion was described by Sir William Gull as a cretinoid condition and by Ord as myxœdema. In dealing with this whole syndrome it is important to keep in mind the fact that hyperthyroidism does not progress gradually and in an orderly manner to the other extreme of myxœdema. An infinity of phases divide the two, and in these sometimes hyperthyroidism, sometimes hypo-

thyroidism appears to predominate. Moreover, there are effects of hyperthyroidism which do not immediately pass away when hypothyroidism gets the upper hand, and so also there are signs of myxœdema which may persist through a subsequent phase of hyperthyroidism. In an early case the picture may be comparatively simple, but in one of long standing the clinical appearances may be complicated not only by the varying effects of previous phases, but by the predominance of symptoms referable to other endocrine organs the implication of which is secondary.

The Clinical Picture of Graves' Disease.

The signs and symptoms of Graves' disease are conveniently summarised by the late Dr. T. D. Savill (1), whose *System of Clinical Medicine* is not quoted because his authority is greater than that of other writers, but because he was more concerned to crystallise the essentials from the very extensive literature on this subject. Dr. Savill summarised the following five chief groups of symptoms :—

(*a*) *Cardiovascular.*—Including palpitation, increased frequency and tumultuous heart action, paroxysmal dyspnœa, cardiac murmurs, sometimes dropsy, and albuminuria.
(*b*) *Nervous.*—Including nervousness, irritability, insomnia, depression, alternating with excitement, mania, vertigo, hallucinations, tremor, sudden perspirations, loss of hair and diarrhœa.
(*c*) *Thyroid Enlargement.*—Variable but always present at some stage. Mechanical effects of thyroid enlargement, such as change of voice.
(*d*) *Exophthalmos.*—Present in varying degree, though sometimes not until late in the disease.
(*e*) *Disturbance of General Health.*—Progressive weakness.

Savill does not mention wasting, but that this is a characteristic feature is acknowledged by most writers. He gives the following additional facts :—

(*f*) Upwards of 95 per cent. of cases are females, a large number young adults between the ages of 15 and 30.
(*g*) Heredity has not been traced, but hereditary neuroses are commonly present.*
(*h*) Fright, anxiety, love affairs, and mental overwork are potent factors in determining the disease.

The same writer details the following as the chief characteristics of myxœdema :—

(*a*) Weakness of very gradual onset with characteristic slowness of action, etc.

* The writer is informed by Dr. Gordon Ward that he has under his care at the present time two families in which mother and daughter have been affected with exophthalmic goitre.

(*b*) Puffy, immobile, vacant face with flushed cheeks. Scanty hair. Brittle nails.
(*c*) Thickened skin.
(*d*) Intolerance of cold. Dry, scaly skin, slow pulse.

From the foregoing quotations we can obtain a sufficiently accurate idea of the main symptoms of the hypo-hyper-thyroid syndrome, but before proceeding to a detailed comparison with nymphomania I will indicate the chief features in a typical case of the latter disease.

The Clinical Picture of Nymphomania.

The disease is most commonly met with in pedigree herds, the individuals comprising which are commonly of a distinctive nervous temperament. In some cases it is hereditary. The subject is always of the female sex, and the usually antecedent circumstances are pregnancy and heavy milking. Owing to the habit of removing the calf from the mother, which is an essential if regrettable factor in the production of milking records, the emotional stress occasioned by these circumstances is exceptionally severe. Those who have heard the continuous cries of the cow from whom the calf has been removed will be able to form some estimate of its severity.

Early stages of the disease most frequently seen during the latter half of pregnancy seldom come under the observation of veterinary surgeons, and when the stage of nymphomania is reached the chief symptoms of hyperthyroidism have been established for some time. The animal is excitable, nervous, and even maniacal. The sexual phase known as œstrum becomes continuous, and the animal will ride others and even inanimate objects. The symptoms are further aggravated by the presence of a bull. This stage, the "nymphomania" of text-books, is of several months' duration in an average case, and often terminates in the slaughter of the animal, since it is no longer of use for milking or breeding purposes. At this stage there can always be found follicular ovarian cysts and rupture of these will ameliorate the symptoms. In the next stage the cow begins to assume male characteristics. The voice changes, there is thickening of the neck and alteration in the shape of the trunk, together with other signs which will be described later. The last stage is one of lethargy and slow pulse, a condition in which the results of previous stages still remain, but no longer progress. Although these stages are described in their most usual order, it is to be remembered that any individual case may be difficult to assign to a particular stage, and that the progress is rather a swing backwards and forwards, with a prevailing general tendency to myxœdema than an ordinary process such as we have described.

XUM

We now return to our thesis: That "nymphomania" in cows is a part of a constitutional disorder known in human pathology as Graves' disease.

To establish this it is necessary to compare the symptoms in fuller detail. It must be explained that every attempt has been made to procure specimens for histological examination. Some have been obtained, but only with very great difficulty, notably from one case of very long standing in which the thyroid was obviously fibrotic. But the specimens are so few, and there is so complete a lack of normal standards in cattle, that the case must for the present stand or fall

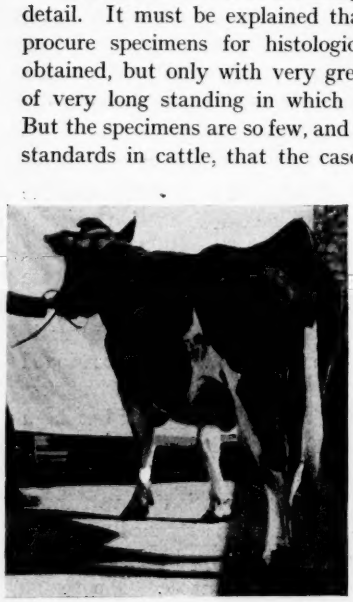

FIG. 2.

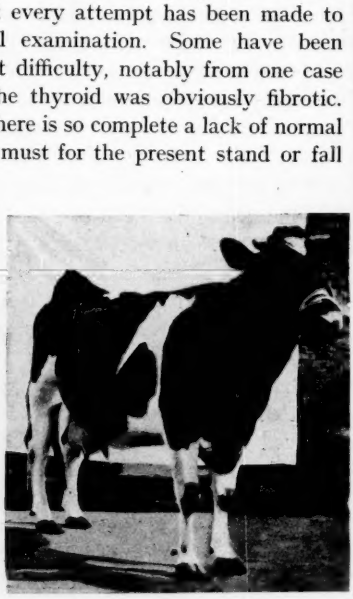

FIG. 3.

These illustrations give some idea of the changes to be seen in the hind quarters of the affected animal. The patella of the right leg became displaced painlessly whenever the beast was turned round sharply.

on clinical grounds. In any case it is not claimed that this paper can do more than afford a basis for future inquiry.

COMPARISON OF GRAVES' DISEASE AND NYMPHOMANIA.

Age and Sex Incidence.—In both, the victims are predominantly females at the fertile period of life. This predominance is 95 to 5 in human beings (1) and as much or more in cattle. No case has yet been described in a bull, but it may be remarked that bulls have a limited commercial value, and find their way to the market at an early age. There may be one bull to twenty cows in the country at any particular time. Moreover, the bull is not exposed to the stresses incidental to heavy milking and pregnancy, but on the contrary is kept quiet and secluded. Finally, it may be noted that diagnosis would be extremely hazardous. For all these reasons we may have

to wait some time for a description of the disease in the male sex in cattle.

Breed and Heredity.—Both occur typically in individuals of "thoroughbred" temperament. The writer knows of the disease in three successive generations in cattle, and is informed of hereditary cases in human families. Whether it is more than inheritance of temperament may be open to doubt, but, on the other hand, it may also be doubted whether temperament is anything more than an expression of the endocrine balance in individuals.

Exciting Causes.—In the human disease exciting causes seem to operate mostly through the nervous system. At first sight we may hardly expect to find an analogy with these in cattle. But the distress of the mother after removal of her calf must be admitted to be a

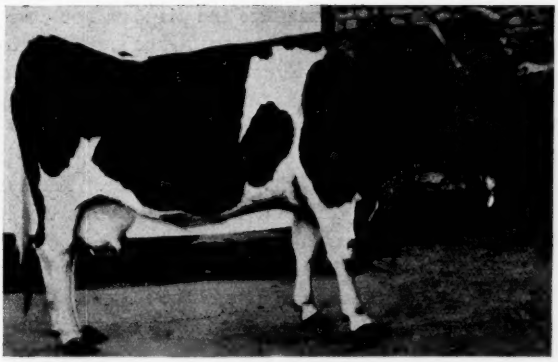

FIG. 4.

Same cow as shown in Figs. 2 and 3 two years later in the final myxœdema stage. Note the peculiar position of the sacrum as a result of the ligamentous relaxation. The external angle of the ilium has been bent down. The thick "bully" neck.

frequent antecedent, and to be an emotional shock greater than falls to the lot of most human cases.

Cardio-vascular Symptoms.—The writer has been able to satisfy himself that the typical tumultuous action of the heart, which may even be audible at a distance, is present in cows as in human beings during the early stages of the disease. Tachycardia accompanies it. It is not possible to identify palpitation in cows, nor have dropsy or albuminuria been noted; but these are not common in any case.

I have under my observation at the present time a cow that developed nymphomania eighteen months ago. She recovered quickly at the time from the sexual mania, but is still affected with tachycardia and extreme nervous irritability.

Nervous Symptoms.—On general grounds the similarity between the two diseases is exact. In both cases we have extreme irritability, and excitement with mania as an occasional *dénouement.* In Graves' disease tremor is most apparent when the hand is held out, or held in such a way that no steadying weight is thrown upon the limb. These circumstances do not occur in cattle. I have noted a fine skin tremor presumably due to the underlying muscles, which may be analogous to the tremors seen in Graves' disease. Diarrhœa is seen in cattle during the early stages, a cow often having frequent evacuations if a stranger appears, or from other slight cause. This reflex is under closer control and is naturally less apparent in human beings, but has nevertheless been recognised and mentioned in the literature, e.g. Savill (1), already quoted.

Thyroid Enlargement.—This is not an invariable feature in women, but is usual, and is easily seen since the gland is not deep seated. In the cow it is deep seated, and consequently it is difficult to appreciate changes. We have no evidence either way that it is enlarged or not enlarged. This makes it doubly unfortunate that no histological reports are available.

Exophthalmos.—This has been observed in the early acute stage of nymphomania and is common in Graves' disease. The eye of the cow is normally somewhat exophthalmic, which makes this sign less easy to detect unless one is on the look out for it.

General Health.—In both diseases failure in this is of the same character, wasting being the most prominent feature.

Myxœdema as a Sequel.—This is well known in Graves' disease, and advanced cases of nymphomania present similar symptoms. The hair becomes dry and harsh, the disposition extremely lethargic, and the pulse slow. I have been able to satisfy myself as to thickening of the skin, but this is naturally less apparent in a normally thick-skinned and hairy animal. The animal becomes constipated. The above parallels between the two diseases amount almost to identity. In each case there is a stage of excitability (the detailed symptoms being in many cases identical), followed by one of lethargy, mental and physical. In both conditions there is an extraordinary type of heart action which is typical and which is by universal consent associated with thyroid hyperactivity. Both present the highly characteristic age, sex, and temperament incidence, and in both there are the possible terminations, namely, cessation of the disease or steady progress to a state of lethargy. That the parallel may be more complete it may here be noted that the administration of thyroid extract has in each case the same effect, namely amelioration of the hypothyroidism symptoms.

XUM

My opinion is that the evidence already produced is sufficient to establish a degree of clinical identity which goes far to justify the thesis already put forward, namely that nymphomania and the Graves'-myxœdema syndrome are essentially the same. It will now be assumed that this is true for the purpose of considering the following symptoms, which are of much greater interest and importance when regarded from this point of view, namely: (1) Sexual excitement—nymphomania as a symptom; (2) pelvic and ovarian disorder; (3) assumption of male characteristics.

Nymphomania as a Symptom.—Œstrum in the cow normally lasts for about twenty-four hours. It seems to coincide with the final period of ripening of an ovarian follicle, and is certainly brought to a conclusion by the rupture of a follicle, whether natural or artificial.

FIG. 5.
An illustration of a case of exophthalmos. This cow has tachycardia, associated with extreme nervous irritability.

I have several times terminated œstrum in this way. The first sign of nymphomania is commonly a lengthening of the œstral period, perhaps to three or four days. This attracts the attention of the herdsman, and if a veterinary surgeon be consulted he will invariably obtain the history that the cow has been one of exceptionally nervous disposition. This history is best obtained by suggesting the contrary, which will be denied. At this time it is always possible to find an unruptured follicle, and if this be ruptured *per vaginam* œstrum terminates and the animal may return to the normal state, or may suffer from a recurrence of symptoms associated with delayed bursting of the follicle at the next œstral period. A repetition of artificial rupture on two or three occasions will not uncommonly restore the animal to normal health. Failing any treatment, œstrum is further prolonged, until it becomes practically continuous and the associated

symptoms increase in severity. The excitability becomes extreme, and the disease, " nymphomania," is fully established.

It does not seem that there is any human parallel to these symptoms, but it has to be remembered that no such parallel has been sought for, and that for social reasons any such symptoms would be kept in the background. The cow does not undergo the years of training which prompt women to suppress manifestations of sexual desire. The result is frank and obvious nymphomania. We cannot expect to see such symptoms in subjects of Graves' disease, nor in fact do we find them mentioned in the literature. Geikie Cobb (2) says, " Perhaps the saddest of the changes wrought by this disease are the changes for the worse in the psyche," but the exact intention of this remark is not certainly evident. It may be that gynæcologists can throw some light on the question. Certainly hyperthyroidism is associated with the attainment of puberty and also with pregnancy, so that we might well expect psychic change associated with changes of a physical nature. But beyond this we cannot go at present.

Gynæcological Disorders.

Pelvic troubles are extremely frequent in cows, occurring perhaps in 40 per cent. in the form of metritis, cervicitis, etc., and are all of a definitely septic type. Such pelvic troubles may of course complicate nymphomania, but they are not sufficiently constant to suggest that they have direct ætiological significance. A constant pelvic abnormality in nymphomania is patency of the cervical canal together with a relaxation of the vaginal tissues. If the cow has calved, there is added definite subinvolution of the uterus. The second and most striking abnormality is the invariable existence of ovarian cysts. These are always follicular in origin, and originate in ripened but unburst follicles. That they are closely associated with the nymphomania is evidenced by the cessation of the latter if they be ruptured or removed by ovariotomy. Their exact mode of action cannot be understood without advancing into the debatable fields of endocrinology, and to do this would unduly enlarge the scope of this paper.

Very few figures seem to be available with regard to pelvic lesions in Graves' disease. Helen Gurney (7) states that in 8 per cent. of cases there was " disturbance of the generative function " ; in 15 per cent. there was amenorrhœa ; 6 per cent. of the cases developed after miscarriage. These figures are neither very definite nor very satisfactory. In a case published by Ward (the reference is unfortunately not obtainable) both ovaries were cystic, and it is probable that isolated cases of the sort might be disclosed by a more thorough search of the literature than active country practice of a pro-

fession allows. But at present there is no real information available either as to the existence of nymphomania or of pelvic lesions.

There remains for discussion under this heading a pelvic lesion of quite different character, i.e. actual pelvic deformity, and at times multiple fractures. There would appear to be at least two factors present, namely, slackness of ligaments and softening of bone. The photographs show the resulting clinical characteristics, the most important of which is the elevation of the root of the tail. This would seem to be due to a rotation of the pelvis on the line joining the acetabula. That the change is due to ligamentous relaxation is strongly suggested by the fact that it disappears under appropriate treatment, whereas bony changes would remain. Relaxation of ligaments is symptomatic rather of myxœdema than of hyperthyroidism. Geikie Cobb (3), in connection with the human disease, says that he is " certainly of opinion that the relaxation of the ligamentous structures in submyxœdema is something more than an artificial symptom. He has noticed it in a number of patients affected with the disease in which the diagnosis was confirmed by treatment. There are certain other signs of ligamentous relaxation which have been noted in cows, e.g. " rheumatic " joint sounds, slipping of the patella, and partial eversion of the vagina. It is possible that similar signs might be found in human beings.

Multiple fractures of the pelvis do not seem to have any parallel in myxœdema. One is naturally inclined to suggest that they are due to excessive decalcification in the hyperthyroid stage, but there is no direct evidence of this. Little is known about these fractures beyond the fact of their occurrence.

Assumption of Male Characters.—This is a very striking characteristic of the middle stages of nymphomania. The nervous irritability of the earlier stages give place to a certain aggressiveness, and the cow will paw the ground, kick out with its forelegs, and charge other animals or its attendants. The voice changes to the deep note of the male sex. The appearance of the head changes together with the expression, and the neck grows thicker. Even those who are inexpert in veterinary matters frequently comment on the change. The female characters regress. The mammary gland atrophies, while the clitoris is often enlarged. The general appearance is well shown in the photographs exhibited.

These remarkable changes do not seem to have any complete parallel in myxœdema, but there is a partial parallel in the loss of many attributes of femininity. It is usually allowed that the anterior portion of the pituitary becomes more active as the thyroid activity fails. But as a rule this is not sufficient to give rise to symptoms of hyperpituitarism in women. Swale Vincent (5) sums up the matter

as follows: "When the pituitary hypertrophies as a result of subthyroidism, there are no symptoms of superpituitarism, so that the pituitary as a whole does not become more active."

On the other hand, the increasing ovarian activity that accompanies the subthyroidism (especially when full sexual indulgence is allowed), undoubtedly stimulates the anterior lobe of the pituitary. But beyond the assumption of male characteristics, there are no other indications suggestive of acromegaly.

There are certain reasons for supposing that the thyroid-pituitary balance in cattle is not of quite the same order as in man. It is a

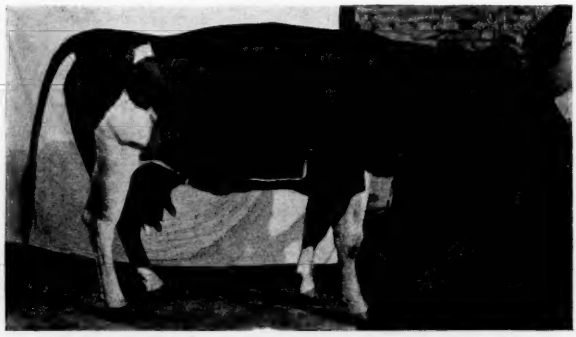

FIG. 6.
The case shown in this illustration is peculiar in that the ligamentous relaxation gave rise to a curvature of the spine as shown. The cow could not keep the tail low enough to touch the vulva.

fact (and to this Swale Vincent (4) also calls attention) that adult herbivora can support life quite well without their thyroid glands; this might perhaps be interpreted as meaning that adult herbivora lose their thyroid at the close of the fertile period of their lives. Cases of nymphomania have not yet reached this stage, but there is no doubt that this disease quickly renders them sterile, and that exhaustion of the thyroid seems to supervene much more speedily and completely than in man. One is consequently not very surprised to find that actual signs of hyperpituitarism dominate the clinical picture in cows, but are relatively little obvious in human beings. But this again is an excursion into endocrinology which may be thought out of place in a purely clinical picture.

CONCLUSIONS.

(1) The foregoing details appear to substantiate the thesis that the so-called nymphomania of cows is a part of the Graves-myxœdema

syndrome, the phase of sexual excitement probably marking the passing over from the hyper- to the hypo-active stage of the thyroid. (In this connection it is interesting to recall Colonel McCarrison's (6) written query at a recent discussion on Graves' disease: "Has suppressed excessive or perverted sexual function anything to do with its onset?" From a purely clinical point of view, excessive coitus in an affected animal tends to force it more quickly into the hyperthyroidic state than the reverse.)

(2) It is further obvious that the whole syndrome from hyperthyroidism to myxœdema can be passed through much more quickly in cattle, and is in many ways more easy to study, since the life of a cow may be terminated at will.

FIG. 7.

The same cow as shown in Fig. 6 after the administration of thyroid extract for two months. The deformity to be seen is due to fracture of both sides of the pelvis displacing the sacrum. The sacro-sciatic ligaments have completely recovered their tone.

(3) For the cattle breeder and the owner of pedigree herds the subject has great importance, because nymphomania leads to sterility, and thus to great commercial loss.

(4) Nevertheless the subject cannot be studied as it should be except on an experimental farm with proper laboratory accommodation, and it is to be hoped that someone to whom these facilities are available will devote the necessary attention to the subject.

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(6) McCARRISON: Discussion on Exophthalmic Goitre (British Medical Association Annual Meeting), see Brit. Med. Journ., November 11, 1922, p. 913.
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XUM

SOME COMMON CAUSES OF POISONING AMONG DOMESTIC ANIMALS.

By G. W. CLOUGH, D.Sc.,
Professor of Chemistry, Royal Veterinary College, London.

Rat Poisons.—The toxic agents commonly employed in rat poisons are phosphorus, arsenic, barium carbonate, and strychnine. The extravagant claims made by some manufacturers appear to have caused a certain amount of neglect of obvious precautions in the use of these dangerous substances. This lack of care has frequently produced damage among domestic animals. For example, a short time ago a large number of birds died on a poultry farm; analysis proved that arsenical poisoning was the cause of these deaths. A rat-catcher had guaranteed that his preparation would kill no animal except a rat, and the farm had therefore been well dressed with this material. A chemical analysis showed that the chief ingredient of this "harmless" mixture was white arsenic. But perhaps the most remarkable claims are made for barium carbonate. More than one hundred and fifty years ago James Watt, of engineering fame, observed that barium carbonate administered to dogs produced vomiting, diarrhœa, and death in a few hours. The present writer has encountered cases of barium poisoning in pigs, poultry, and dogs. In one of these cases a number of pigs, after consuming their food, showed symptoms of poisoning—vomition, staggering, and paralysis; ten died in a few hours. Analysis revealed the presence of barium in the stomach contents, and a sample of the food was found to contain barium carbonate, the source of which was a rat poison. It is thus evident that barium carbonate is a deadly poison, notwithstanding the fact that it is not included in the Schedule * of Poisons of the Poisons and Pharmacy Act. I have before me a tin, on the label of which is printed a "Certificate and Guarantee: We hereby certify the contents of this tin to be (1) Free from Poison and Harmful Substances; (2) Safe to use under any condition." The active ingredient of the contents of this tin is barium carbonate.

In connection with the subject of rat destruction, it may be of interest to note that during the past two years ships have been fumigated with hydrogen cyanide in order to destroy rats and vermin. The United States quarantine regulations of 1920 require vessels engaged in trade with foreign ports to be fumigated twice a year, and it is therefore necessary to fumigate the passenger quarters of trans-Atlantic liners. Hydrogen cyanide was indeed used in 1890

* The Schedule does not, of course, include all poisons. Moreover, it is not definite, thus "all poisonous vegetable alkaloids" and "all poisonous cyanides" are scheduled poisons.

in the orange orchards of California and Florida, and in 1898 it was used by the Cape Government railways for vermin in sleeping carriages. Since the war hydrogen cyanide has been extensively employed in American, Australian, and Italian ports. Fumigation with this agent is at present being carried out in this country by the Cunard and White Star Companies. It seems probable that this highly toxic vapour will come into general use for large scale destruction of vermin, but it is obvious that elaborate precautions are necessary, and that the work should only be prosecuted under expert direction.

Medicine.—The widespread faith in the "secret remedy" has made the manufacture of proprietary medicines a lucrative business. The manufacturer's knowledge of the effect of advertisement, coupled with his little knowledge of the physiological action of drugs, has not infrequently made the patent medicine a very dangerous thing. A farmer gave to each of six ewes a certain "Ewe Drench," and all of these ewes died within six hours. An analytical examination showed that the drench contained nearly 95 per cent. of zinc sulphate. Antimony compounds are still largely used in proprietary veterinary medicines. The writer has frequently found antimony present in viscera from horses and sometimes in pigs, goats, and dogs suspected of having been poisoned. There can be little doubt that in some cases antimony has been the cause of damage.

The risk attending the use of anthelmintics may be exemplified by reference to two recent cases. A dog breeder administered an advertised worm mixture to a litter of five spaniels ("very strong and healthy pups"), a litter of nine Sealyhams nine weeks old, and also to a retriever ten weeks old. The five spaniels, five of the Sealyhams, and the retriever all died after becoming ill on the day following administration of the medicine. The mixture was found to consist of santonine and castor oil. Three bull mastiff pups seven weeks old were each given a worm ball, advertised to be the most certain cure for worms in dogs. They all died within twenty-four hours. Analysis revealed the presence of santonine, areca and castor oil in the balls. It would thus appear that santonine is a dangerous drug, and that it is not wise to prescribe it for young dogs. It also seems desirable to avoid strychnine in dog practice. Cases have come under the notice of the writer in which doses of only $\frac{1}{120}$ of a grain of this alkaloid have produced the usual symptoms of strychnine poisoning in dogs, some of which did not recover.

Food.—It is advisable that the purchaser of a foodstuff should be acquainted with the relative proportions of fat, protein, and carbohydrate in the food, so that a proper balance of these constituents may be maintained in a ration. Owing to imperfect extraction or

other causes cakes are occasionally found to contain what must be regarded as an excessive amount of oil.

Poisoning by the gross adulteration of food is fortunately comparatively rare at the present time. The judicious use of fishmeal as food is often a valuable aid to nutrition, but the quantity of this nitrogenous material should be carefully regulated. Samples of some inferior lots have been recently encountered. Some of these contained an excessive amount of salt, whilst others were largely contaminated with sand. Injury to poultry and pigs has been caused by some of these inferior fishmeals. Ducks appear to be even more liable to salt poisoning than fowls, and this is doubtless due to the fact that ducks are more indiscriminate feeders. After feeding on a mixture containing 10 per cent. of fishmeal, which on analysis was found to contain over 20 per cent. of salt, 60 out of 300 ducks died. In connection with ricine poisoning, due to the presence of castor seed in foods, reference may be made to a recent action in the King's Bench Division (Pinnock Brothers *v.* Lewis & Peat, Ltd., February 7, 1923). The plaintiffs, having bought from defendants a quantity of East African copra cake, resold the cake to B., who in turn sold part of it to another firm, which manufactured that part into cattle food and resold it to a farmer. B. manufactured the remainder into cattle food. All the cattle to which the food was supplied became seriously ill, and on analysis it was found that a considerable quantity of a poisonous substance, castor beans, had been mixed with the copra cake. Judgment was given for the plaintiffs for £550 and costs.

Damage amongst animals has been caused by metallic lead in brewers' grains, barium carbonate in foreign sharps, an excessive quantity of boric acid in sausages, and by arsenic and picric acid respectively in water.

Amongst industrial workers lead is the most frequent cause of poisoning. The labours of works chemists and of Home Office experts during recent years have produced a very striking diminution in the number of cases of industrial lead poisoning. Lead is also the commonest poison affecting cattle, and a large number of other animals are poisoned each year by lead or its compounds. The aspects of lead poisoning merit more consideration than it is possible to give in the present communication.

AN OUTBREAK OF CHICKEN CHOLERA.*

A Bacteriological and Hæmatological Study.

By A. KNYVETT GORDON, M.B.Camb.,

Medical Superintendent of the Virol Pathological Research Laboratories; Pathologist to the Great Western Railway Fund.

In June 1922 I was asked by Dr. H. A. Lake,† of Beaminster, to investigate an outbreak of disease in a chicken farm in his district. The sequel presented some interesting features. The farm in question was devoted exclusively to the rearing of a fine strain of white leghorn fowls which had been bred on Mendelian lines. Previously healthy, the flock had just been visited by an outbreak of disease, the main feature of which was the suddenness of the onset and the fact that—so far as could be ascertained—the chickens mostly died within twenty-four hours with no symptoms except collapse; diarrhœa and catarrhal signs were both absent. Some dead chickens had been previously examined by a lay expert and stated to have suffered from coccidiosis, the diagnosis having apparently been made simply on the presence of white patches in the walls of the intestine. Later I was shown similar birds when I visited the farm; the white spots were obviously enlarged Peyer's patches.

In the first instance, however, I received a package containing six birds, all of which had been alive when despatched twenty-four hours previously from the farm. On arrival two were alive (A and B) and four dead (C, D, E, F). The living birds appeared healthy, but the dead ones were greatly emaciated. A and B were killed and all six birds submitted to a bacteriological and histological examination, including a study of the heart's blood.

Macroscopical Examination.—All showed signs of septicæmia, slight in A and B and well marked in the rest. The pleura and peritoneum were studded with petechial hæmorrhages and the liver and spleen were engorged and soft. The blood was fluid and "tarry," and the intestinal mucosa injected with a few capillary hæmorrhages in C, D, E, and F. No gross sign of coccidiosis was present, and films from the intestinal contents showed no coccidia in any stage. Cultures were taken with aseptic precautions, and films prepared from the heart's blood and spleen of all the birds. Sections were made of the livers and spleens of A and F.

Microscopical Examination.—The livers showed dilatation of the interlobular veins, some biliary engorgement, and fatty degeneration of the hepatic cells, the changes being slight in A and well marked in F. The spleens showed marked engorgement with cells, which

* From the *Lancet*.

† Dr. Lake is also a Member of the Royal College of Veterinary Surgeons.

were found in films stained by the panoptic method to consist mainly of leucoblasts and cells of the lymphoidocyte type.

Bacteriology.—From the heart blood of A and B the bacillus of chicken cholera (Pasteurella) was grown in pure culture. The same organism was also present in cultures from the dead birds, but contaminated with a long sporing bacillus, the nature of which was not fully investigated.

Hæmatology.—This was interesting. I may remark that in the normal fowl the red cells are nucleated and resemble something between the normoblast and megaloblast in the human subject. Polychromasia is absent. The normal leucocyte is of the leucoblast type—i.e. in man the ancestor of both granular (polymorphs) and non-granular cells (lymphocytes). This, incidentally, is what we should expect on the principle that the development of the individual is a recapitulation of that of the race.

In the dead birds the red cells were of three types: (*a*) the normal erythrocyte, (*b*) cells exactly resembling the human megaloblast, and (*c*) large polychromatic megaloblasts, some of which seemed to be intermediate stages between (*b*) and lymphoidocytes. In birds A and B a few megaloblasts were found on exhaustive search, but no polychromasia. The white cells showed total leucocytosis slight in A and B, well marked in C, D, and E, and extreme in F. The cells were mainly of the normal "leucoblast" type, but in all typical lymphoidocytes (the ancestor of all blood cells, both red and white) were present. Of these, in A and B a few only were found on careful search, in F they numbered 25 per cent. The blood of all the dead birds also showed granular masses of protoplasm similar to those seen in advanced human leukæmia; they are probably dead leucoblasts in each case. All films were stained by the author's modification of Pappenheim's panoptic stain.

I then visited the farm with Dr. Lake. It was obvious that with the exception of one open field all the ground adjoining the huts in which the fowls were kept must have been by this time heavily charged with excreta of infected birds. It seemed impossible to devise any method of adequate disinfection thereof, so even if all the birds were slaughtered a recrudescence of infection in the new stock seemed likely to occur. Consequently, a vaccine was prepared from the strain of Pasteurella isolated from birds A and B and the whole colony of 700 birds was inoculated therewith, an initial dose of 1,000 millions being followed a week later by double the quantity. The result was successful. With the exception of four birds, which died shortly after the first inoculation and were probably infected, all recovered, and no further cases of disease occurred. As much

time and research had been spent on the perfection of the strain, which was a valuable one, the result was regarded as satisfactory.

While vaccination with attenuated live cultures is well known as one of Pasteur's classical experiments, I have been unable to discover in the literature of the subject any similar case of the use of an autogenous dead vaccine in chicken cholera. As the disease appears to be fairly prevalent I suggest that this procedure may be of practical use to chicken farmers. To the hæmatologist the presence of lymphoidocytes in the blood of the infected birds affords an interesting study in comparative pathology. In the fowl—as in some reptiles—the lymphoidocyte is only one stage further back than the normal leucocyte, so one would expect this cell to be present in the same way as in man when under certain conditions—e.g. severe bacillary infection—leucoblasts appear in the circulation. I may here remark that in a study of the blood in very young tadpoles I have found lymphoidocytes present together with intermediate forms both between the nucleated red cells and the white cell of the blood of the fully developed frog. Here, as in birds, this is of the leucoblast type.

I must gratefully acknowledge the courtesy of the owner of the farm, Mr. C. A. C. Streatfield, who throughout the investigation gave me every facility, including detailed clinical notes of the epidemic.

Clinical Articles

POISONING FROM CASTOR BEANS.

By Major-General Sir JOHN MOORE, K.C.M.G., C.B., F.R.C.V.S.,
Late Director-General, Royal Army Veterinary Corps in France.

During the war a serious instance of poisoning from castor beans occurred in a unit of the British Expeditionary Force, France, resulting in the death of several horses and illness of a considerable number of the unit. Very prompt action was taken by the veterinary officer in charge and the D.D.V.S. of the Army. There was no mistaking the cause, as the oat supply of the unit was heavily admixed with castor beans. No doubt the vessel which transported the oats in bulk had previously carried castor beans as part cargo, and in the hurry of requirement had not been properly cleaned out. The unit affected had received the final sweeping up of the hold. The unissued balance of the vessel's contents instead of being wasted was handed over to veterinary units on lines of communication, as they were in a position to effect screening and cleaning of the oats.

Another instance of poisoning traceable to castor occurred at Marseilles from two transport horses gaining access to ground-nut

cake while on station duty. It is quite easy to understand adulteration of Marseilles cakes, or at least to suspect it, as mills used for crushing of various seeds may include castor.

ACUTE LEAD POISONING IN A MARE.

By R. GREGG, M.R.C.V.S.,
Bakewell.

Subject.—A four-year-old shire mare, unbroken, in foal, and in excellent condition.

History.—Called to this case on January 18, 1922. On arrival at the farm I was shown this mare in a box, and was presented with the following particulars by the owner :—

The mare was one of three which had been running at pasture all winter, and it was his custom to take these horses a daily ration of hay. This day in question all three started feeding as usual when they received the hay, but this mare, after eating greedily for a few minutes, stepped back, poked out her nose, and started to salivate very freely. After watching her for a few minutes he removed her to the stables.

Symptoms.—The animal was standing looking rather dull and miserable, with head poked out and saliva dribbling from her mouth. Temperature normal, pulse slightly accelerated, mucous membranes normal, and respirations about 20 per minute. There was a slight fullness along the cervical portion of the œsophagus, the manipulation of which the mare seemed to resent.

Diagnosis.—Plugging of the œsophagus with dry hay, and treatment was directed accordingly. About 3 ounces of Ol. Lini. were given, the head tied up, and the part massaged and fomented.

Seen next morning. All symptoms of choking had disappeared, but the mare seemed more depressed. Examination revealed a temperature of 105° F., pulse 80, respirations 30, and mucous membranes intensely injected.

I was now somewhat at a loss to explain these, but ordered the mare to be rugged up, etc., and prescribed quinine electuary, alternated with Pot. Chlor. and Belladonna electuary.

About 7 p.m. on the same evening, at the request of the owner, I visited the animal again, and learned that she had shown signs of colic several times during the day. The attacks lasted varying periods, but only once did she lie down, and she had some difficulty in rising again. Each time, according to the owner, she showed weakness in the hind quarters. In the intervals between the attacks the mare appeared fairly normal, and would drink a quantity of chilled water. I found the mare in the depth of an attack, the most prominent

symptoms being as follows: Laboured and quick respirations, pulse quick and running down, patchy perspiration, mucous membranes injected, temperature 106° F., twitching of the superficial muscles, and partial paralysis of the hind extremities, most marked when movement was attempted. The hind legs gave one the impression that they were too short for the body, due to flexion of the joints. Left undisturbed, the mare stood as if rooted to the ground in front, but kept constantly paddling behind, and when forced to move, control of the fore parts seemed normal, but great difficulty in getting the hind legs forward was manifested, and the hind quarters swayed from side to side. The abdominal pains seemed of a sub-acute nature, the head being turned towards the flanks and the animal crouching somewhat. Peristaltic sounds were absent. I proceeded to make a rectal examination.

The mare had only passed fæces once from the night previous, and these, together with what I obtained on rectal examination, were rather dark in colour and somewhat stiffer in consistency than one expects to find in a pasture-fed animal.

Just as I finished my rectal examination the animal dropped and died in a convulsion.

Post-mortem.—Acute congestion of lungs, trachea, and larynx. Greenish grey appearance of the intestinal mucous membrane. Liver greyish, soft and swollen, and lymph gland congested.

A portion of liver and ingesta were sent to W. H. Roberts, M.Sc., F.I.C., City Analyst, Liverpool, for analysis, together with history and symptoms. He reported as follows:—

"The ingesta contained $\frac{1}{3}$ grain of lead per pound and a trace of zinc. The liver contained $\frac{1}{8}$ grain of lead, and $\frac{1}{5}$ grain zinc per pound.

"From analysis and symptoms I am of the opinion the mare died from lead poisoning."

I have since learned that two horses died rather suddenly on this farm inside the previous few months, one of which had been a decided roarer and doing badly. Also a cow died a year previously showing signs of epilepsy, but with the exception of these cases nothing suspicious can be remembered.

This land has all been mined for lead, and one can see the old shafts in every field; also it may be mentioned that the water supply is from the land itself.

Perhaps the explanation of the fact that nothing suspicious occurred until recently may be that until two years ago the stock was not exclusively confined to this one farm, but allowed on to another farm (which is free from lead) for part of the season. Nevertheless,

XUM

it seems peculiar that of the three young horses mentioned under the same conditions one should be taken and the others left.

AN UNUSUALLY LARGE ELBOW TUMOUR.

By JAMES D. FULTON, M.R.C.V.S.,
Melrose.

Subject.—Clydesdale gelding of about ten years used for timber hauling. The animal was bought about two years ago, and had a capped elbow at that time about the size of a small coco-nut. The tumour grew steadily until its removal about a fortnight ago, when it was as big as a full-sized soccer ball. It did not interfere with the horse's working ability, although occasionally it would burst and discharge a foul-smelling sanguineous pus. The owner spoke to me about some two months ago, and asked me if it might be taken off successfully. I replied "I hope so."

Mode of Excision.—I got two assistants to pull tumour towards them so as to stretch the skin at the neck, and I then applied an elastic ligature of motor cycle valve tubing. I wound this ligature round two or three times tightly, and tied it in a single knot. I saw the horse four days later; he was very comfortable, but no signs of cutting had taken place. In or about seven or eight days the skin at the neck was completely cut through its circumference, say, about 14 inches. I removed the ligature and re-applied a fresh piece. In another week it had cut through several inches. Again the ligature was taken off and a third one applied as tightly as before. The owner dressed the cut surface twice daily with common salt solution about 10 per cent. At the end of the third week the neck was about the thickness of a child's wrist. At this stage I endeavoured to twist the tumour off, but found that it had a very much greater hold than it looked. I decided to put another ligature on and allow the tumour to fall off on its own. This happened, strangely enough, the following day. Altogether it took twenty-nine days.

The tumour was weighed, and found to be just over 26 pounds, and it was too voluminous to go into an ordinary stable bucket. The wound surface, having been dressed twice daily for the best part of three weeks, was a particularly healthy granulating one. I have seen the animal twice since, and the raw surface is healing very quickly, there being apparently no pain.

Treatment to the raw surface consists in the application of cold water from a hose, common salt solution 10 per cent. and dry dressing.

The tumour was sent to the Royal (Dick) Veterinary College, Edinburgh, and Professor Mathieson kindly informed me that its structure was that of a fibroma.

The tumour contained a large number of very large bloodvessels, and it had also developed its own nerve supply. One nerve in particular running through the centre of the tumour was as thick as a pencil.

CAKE POISONING.

By R. HUDSON, F.R.C.V.S.,
Retford.

THREE four-months-old calves were fed on milk, mown green food, and a well-known firm's cake nuts.

They had been taking the nuts from the same bag for some days. On one particular day they were given the nuts in a trough, and being short of milk the man took them some warm water from the milk cooler and gave it to them as they finished the nuts. Shortly afterwards he returned to them with some green food, and was surprised to find them all blowing hard.

Arriving about two hours after they were found amiss, I found one dead. The others had bloody froth escaping from mouth and nose, breathing with great rapidity, œdematous swelling about the limbs, from which bloody serum was oozing. All were tympanitic, but not sufficient to produce suffocation.

Post-mortem examination showed congestion and œdema of the lungs, fermentation taking place in the rumen, and no sign of gastro-enteritis.

The food was analysed and found free from detectable poison, and the remainder was afterwards used with safety.

The other two calves recovered. Was the poison Hydrocyanic Acid produced by fermentation?

HYDROCYANIC ACID POISONING IN YOUNG CALVES.

By P. WILSON, M.R.C.V.S.,
Lanark.

SOME time ago, about 7 a.m., I received an urgent message to go to W. K.'s farm, as he had found one calf lying dead, all the others showing some signs of illness, and one or two of them quite like dying also. On reaching the farm, the dead calf was lying at the gate, and as its death had already been reported to the County Authorities, I did not touch it.

A little farther in the field a calf was lying sheeted up. The symptoms shown by this animal were: subnormal temperature, very weak pulse, very weak and shallow respirations, general insensibility, inability to hold the head up, if the head were raised it dropped like an inanimate object when it was released, the eyes were fixed and staring, and there was nearly, if not quite, total blindness.

The others were all moving about, and the owner said that when they were first noticed to be ill, they had a tendency to go backwards, with the legs straddled, and the head elevated, and to fall over on to the side.

Asked as to the food, the owner said he had just begun to use the second of two bags of ground linseed cake, which he had bought; that it was prepared by soaking with cold water and allowing it to stand between meals, when it was made down into a drink, and given to the calves. Nothing happened during the time the first bag was used. The trouble was diagnosed as hydrocyanic acid poisoning.

Treatment.—The calf that was seriously ill was given ʒij Æther subcutaneously immediately, and the hot cloths, which the owner had already wrapped round the calf, were renewed.

When this had been done, we went to have a look at the others. The older and larger calves appeared to be all right; the smaller ones were dull in appearance, moved in a stiff manner, and showed no inclination to feed; but after watching them for a short time, one concluded that the effects of the poison were passing off, and that no treatment was necessary.

On going back to the first one, it was showing some signs of improvement, notably it could support the head from the ground for a little at a time, and could evidently see a little also. Some sweet milk was now got, and as the calf could swallow when drenched, it was given a dose of Spt. Ammon, Aromat. Æther and Fl. Ext. Nux Vomic. in a little milk. The hot cloths were renewed. Two doses of Spt. Ammon. Aromat. and Æther were left, with instructions that the first was to be given in an hour, and the other two hours after the first, both to be given in milk, and the hot cloths to be renewed occasionally until the calf rose; it was then to be thoroughly dried, rubbed with a little thin mustard and water, and dry sheets put on.

On visiting the next morning I was told that the calf got up about twelve noon and walked into the house. It was fed on porridge and milk for some time, and did well. The others also had a change of diet, and did well. But it was nearly a week before all symptoms of ill-health passed off.

The dead calf and the one worse affected were the two youngest calves, and were about five months old. The dead calf was found in the morning lying where they were fed the previous night, from which place it had never moved away.

The others were noticed to be ill in less than a quarter of an hour after they were fed in the morning. Hugh Begg, Esq., F.R.C.V.S., County Veterinary Inspector, certified that the cause of death was not anthrax.

Each calf was receiving about 10 ounces of the meal twice a day, made into a gruel as stated before.

I had a sample of the meal analysed, and it was said to contain 0·035 per cent. of H.C.N.

Professor R. G. Linton, to whom a sample of the meal was sent, also had it analysed, and it was found to contain 0·04 per cent. of H.C.N. Considering the volatile nature of the poison, the discrepancy is not great between the two analyses. They give about 1·6 and 1·8 grains of H.C.N. respectively in the amount of food given, but as the poison is volatile, a certain amount would be lost, so that it might be taken that each calf received about 2 grains H.C.N. in its food, and this amount must be looked on as the minimum fatal dose for a five-months-old Ayrshire calf, as calves a week older did not succumb.

NOTES *RE* LEAD POISONING.

By E. P. EDWARDS, M.R.C.V.S.,
Mold.

LANDER in his *Toxicology* says: "Lead poisoning of cattle almost always results from eating white paint or red lead," and he makes no mention of cases arising both in horses and cattle in lead-mining districts from ingestion of the mineral galena (PbS) or its salts.

He further says: "The relative tolerance displayed by the horse is a remarkable point, and is on a parallel with the comparative rarity of cases among those animals."

The late Professor W. Williams, in his *Veterinary Medicine*, says: "In the horse roaring is considered in Wales the first symptom of lead poisoning, and in the lead districts about Holywell and Flint every roarer is said to have had lead." Without going nearly so far as the popular belief, my experience over a long period in this district is that horses, and especially young animals, are very susceptible to the poison, and that it produces all degrees of an incurable roaring.

In this article, however, I wish more to direct attention to some points in relation to lead poisoning other than those of treatment or symptoms, but concerned with the special sources and distribution of the poison in lead districts, and with the jurisprudence of the subject. Some large lead mines exist in my district, and cases are by no means infrequent, and the distances from the actual mines, where cases are met with, are sometimes surprising.

Moreover, in one part of the district there is a lead-smelting works, and as it is a factor quite distinct, both as to range and character of distribution of the poison, it will be noted separately.

Broadly speaking, the cases of poisoning in lead districts arise through :—

(1) Contamination of streams, rivers, or pastures, with the effluent silt or mud from the washing pits.
(2) Contamination of pastures from the dust of waste heaps, or dumps, containing unrecovered lead.

When I use the word "lead" here, I mean the virgin mineral "galena," which is PbS, with varying proportions of "blende" (ZnS).

Now, PbS is quite insoluble, and a very stable chemical combination, but nevertheless it is the source of every case of lead poisoning in cattle and horses I have met with here.

The mineral is crushed and ground at the mines, and the separation of the metals from the rock, more or less imperfectly performed by the processes of "sliming" and "silting," in which enormous quantities of water are used, and it is not too much to say, that however great care is taken by the proper use of "catchpits," this effluent water always contains some percentage of lead-bearing slime.

Again, at lead mines enormous dumps of this waste slime exist, and heavy rains wash it away on to pastures, on to the roads, and ultimately into some local rivulet, stream, or river. In hot, dry weather these huge dumps become quite powdery, and high winds carry the poisonous dust over neighbouring pastures.

In the case of some small "tributing" mines, the dumps are near the banks of a river, and if a heavy flood comes, enormous quantities may be washed away, and pollute pastures and river beds for long distances, and in the case of the river render it dangerous for several years.

In the spring of 1890 a heavy flood occurred, and part of such a dump was carried away, and large tracts of land flooded. Within a few days of the subsidence of water, and the return of animals to the pastures, I had five cases of acute lead poisoning in cattle, three at one farm, and they all died.

Later that summer, when the river was very low, cases of subacute and chronic poisoning in colts began to come in, and some occurred on land through which the river ran, at distances of three and four miles from the mine. Moreover, these cases in horses appeared again, though in decreasing numbers, for two years after, and the sediment in the summer pools of the river still yielded lead on chemical test.

Here may I emphasise two marked differences in the incidence of the poisoning of horses and cattle in such circumstances :—

(1) The cases in cattle come chiefly *from ingestion of the silt on the pasture,* and even large, coarse, glistening particles of galena are often found in their rumens.

(2) In horses such ingestion is rare, and the cases occur later,

XUM

when the river is very low, and are most often chronic cases of the " roarer " type, and arise *from drinking in the pools of the depleted river.*

But, since the cattle also *drink* in these same pools, it may be asked why they are not as dangerous to them also. The answer is a natural history one, and arises from the difference in the habits of the two species when proceeding to quench their thirst.

The cow walks daintily into the pool, hardly disturbing the river bed, puts her lips to the clear water, and takes her fill genteelly. The horse, on the other hand, walks in with a clatter and splash, and proceeds to paw the river bed until he has made it " soupy " enough to suit his fancy, plunges his muzzle into the muddy mixture, and takes a dose of the deadly silt.

If this is repeated several times a day, there is trouble coming before long.

The most dangerous time for horses to graze on pastures through which a contaminated river flows is when the river is very low, and numerous quiet pools exist in it. When the river is full, and the current swiftly flowing and deep, there is less likelihood of the horse going into the stream; he drinks from the edge, and even if he does indulge in his pawing propensities, he cannot stir up so deadly a mixture as he can in a quiet pool of almost still water. The more level the valley through which flood water runs, the nearer to the mine will be the greatest contamination of the river bed or the pasture, on account of the specific gravity of the toxic mineral, but I can imagine a rush of flood water along a narrow, well bounded gorge carrying it for great distances. As illustrating the distances from the mines at which a river bed may be polluted, I give the following facts: In August 1916 I diagnosed lead poisoning of subacute form in a valuable cart mare. The Alyn River flows through the farmer's yard and meanders through most of his pasture land; adjoining him another farmer further from the nearest mine had five colts, all roarers, and another nearer the mine had two affected.

The latter farmer is, allowing for the windings of the river, some three to three and a half miles from the nearest mine, and the end of the land of the furthest victim is quite six miles away. I took samples of mud from the river bed at intervals beginning within two miles of the mine, and continuing to a boundary eight and a half miles from it, with the result that lead was found in every sample, decreasing in amount at the longer distance, but in sufficiently toxic quantity, and I have no doubt the pollution extended much farther.

In 1917, despite warnings, other cases in horses occurred along this tract with an over-confident farmer who turned colts out on the land in the summer. Legal action was mooted in these 1916 cases,

but as the effluent of three mines was going into this small river, the fixing of responsibility was difficult, and to sue the trio would have been a costly and protracted business, so none was taken.

A case which came before the Courts, and illustrates another way in which the mineral galena proves fatal, is of interest. Some of the waste rock mined is not payable ore, and is sometimes used for road mettle, and is got for the carting away.

A certain Rural Council used this material for a road, part of which consisted of a pretty steep and long hill, which, at a particular point, branched into two roads, at a "letter V angle." At this angle was a gutter, or watercourse, to take any rush of storm water coming down the steep hill, and such water passed through an open culvert into my client's pasture. Months after the repair of this road several heavy rainstorms took place, and shortly after I was called to see three cows belonging to the client indicated, and they all died of acute lead poisoning, as proved clinically by post-mortem and analysis.

Samples of soil, sods, and deposited silt showed the presence of quantities of galena, some visible to the naked eye, and the silt, on chemical test, was rich in lead. Legal action followed, my client won his case ; the Council appealed, and he lost his case.

My client, in addition to farming, kept two teams engaged in carting galena from the mines to the railway ; he admitted that the wagons stood in his yard, and that leakages of the powdered mineral might escape on to the yard, so the Judge allowed the appeal on the ground that this might be one source of the poison, and he declined to fix responsibility on the Council alone.

This decision is open to grave question, in view of the fact that my client had been engaged in this cartage of lead for many years without ill result, that it is inconceivable the cows would eat the farmyard refuse, and that there was ample evidence of lead pollution of his pasture following the use of the mine rock as road mettle.

Still, it is a case which serves to illustrate the subject of the distribution of the poison, as well as that of the uncertainties of the law when possible *contributory* sources of pollution are present. In 1916 I diagnosed lead poisoning in a case two miles away from the nearest mine and far away from possible river pollution, and was much puzzled until I discovered that "mine mettle" was being used to make concrete in the construction of the Birkenhead water-pipe track, which ran through my client's land. The case was easily proved, there was no possible contributory cause here, and the contractor paid without a fight, and also paid "consequential damages"

for land contamination—a point which should never be overlooked in assessing claims in such cases. Respecting my earlier reference to the distribution of lead by a smelting works, I record the following facts :—

In 1913 I was engaged, in conjunction with my neighbour, Mr. W. Hughes, Caerwys, in the investigation of the cause of the loss of several hundred sheep on the estuary of the Dee, which was alleged to be due to lead or zinc poisoning, or both, from pollution of the River Dee by effluents from the large works of a firm who manufacture corrugated sheeting. The alleged poisoning was refuted, and the invasion of various intestinal and lung parasites established as the cause of death.

But the point of interest here is, that owing to the vital importance of the issue to the owners of the huge works at Queen's Ferry, it was necessary to prove up to the hilt that they were not polluting the Dee, and very exhaustive researches by well-known analysts were undertaken with this purpose, with the following results :—

Travelling from Queen's Ferry, samples of sods were taken from land adjacent to the river at intervals of about half a mile, going north toward the site of the smelting works I have mentioned, and quantitative analyses made. The distance to the works " as the crow flies " would be about nine miles, and it was found that the percentage of lead increased in a steady ratio for over half that distance, and then gradually declined, these features being accounted for by there being a maximum and a minimum point, at which subsidence to earth of the poisonous content of the effluent fumes of the chimneys would occur.

Lead and zinc had been found on the margins of the Dee near the corrugated iron works (and also in non-toxic traces in the sheep), and these investigations (in conjunction with analyses of actual effluents from the works) satisfied the Judge who tried the case that the source of such metallic deposits was the smelting works about nine miles away. The case on appeal was taken to the High Court, where the above judgment was confirmed, but must have cost the price of many thousands of sheep.

As to the chemical side of the question, how galena, an insoluble mineral, of very stable chemical character, and containing a metal which produces so few soluble salts, can produce when ingested both acute and chronic lead poisoning, I consider that more research is required—at any rate, than I am aware of.

That *metallic* lead in the presence of water, Co_2 and O, and especially if nitrates are present, produces either soluble salts or salts which are soluble in acids, is well known, but the stubborn PbS may need more potent agencies.

That a number of acute cases may occur together within a few days of the ingestion of the silt from a lead-mining dump shows either that the silt contains lead salts in the form of carbonate, oxide, or chloride, or that the digestive processes are potent enough to produce a soluble salt direct from PbS.

I believe the former to be the correct explanation, and that the exposure to moisture, air, and CO_2 reduces the PbS *whilst in the dump* to oxides and carbonates, which by the action of the gastric juices form the sparingly soluble chloride.

A word as to symptoms. In the cow it may be that you are called to a case of choking. In the first of the batch of cases referred to as occurring in 1890 that was actually the message received, and it simulated a choking case to perfection. So much so, that I put the gag in the cow's mouth preparatory to passing the probang, but when from excitement the animal fell down in a typical epileptic fit, I "tumbled," and examination of the rest of the herd revealed the other incipient cases.

Other symptoms in the cow are digestive disturbance, stubborn stasis of the digestive tract, extreme dullness and lassitude in some, but partial or complete blindness, great excitability accompanied by intermittent fits in others, the latter features being much the more usual. In the horse acute cases are much rarer, and I only differentiate what may be called subacute and chronic cases.

The subacute case is interesting and peculiar. When at rest and undisturbed the animal appears in normal health, but trot him, walk him some distance, or excite him by the threat of a stick, and quite suddenly he develops such rapid and extreme symptoms of dyspnœa that he may even fall to the ground, "gasping," "whistling," and then "screaming" for breath, and may even die from suffocation if the excitement is persisted in.

This train of symptoms has been well named "laryngismus stridulus," so "strident" is the note of the scream for breath on extreme occasions. The chronic cases are those exhibiting little but varying degrees of "roaring" or "thick wind" on exertion or test, and the group of symptoms appear to be due to varying degrees of nervous disturbance.

I have seen two very chronic cases in old ponies exhibiting labial paralysis and dropped ears; in these there was no disturbance of respiration, and in one of them for the only time was I able to recognise the so-called "blue line." In my experience treatment is of no avail, but some of the older animals, if not bad roarers, may recover moderate usefulness in time.

XUM

AN UNUSUAL CASE OF LEAD POISONING IN CATTLE.

By HARRY P. STANDLEY, M.R.C.V.S.,
Norwich.

WHILST my uncle, the late Mr. T. E. Auger, was in practice at Wymondham, on one farm he had to attend some cases which assumed a very mysterious and baffling character. Whenever this client's stock were turned out on one particular field, it often happened that one or more of them were seized with sudden and unexplained illness, and as they all showed acute gastric disturbances only one cause could be assigned to the illness: that it was something the animals must have eaten whilst on the pasture. Once the animals were taken ill they invariably died, and Mr. Auger's client got so dissatisfied that he dispensed with his services and employed another neighbouring practitioner. However, this latter gentleman had no better successes than Mr. Auger, and was quite unable to assign any cause to these cases. After a time the client came back to Mr. Auger, but the cases still went on. The last of these cases occurred after a very high wind, and on going over the pasture Mr. Auger picked up some white pieces of sailcloth, and on inquiry what they were, and where they came from, was informed they had blown off the sails of an old disused windmill which stood on this field. The cause of the trouble was then pretty clear: these pieces, which turned out to be pieces of cloth which covered the mill sails, and had been repeatedly painted, were absolutely saturated with white lead, and on opening the last animal which died, a good number of these pieces were found in the stomach. The old mill was dismantled, and no further case has occurred on the farm since.

About two or three years ago I was requested by one of my assistants in a branch practice to go over and meet him in connection with several bullocks which had been found dead on a farm, and some more which were showing symptoms of illness. The dead bullocks had already been taken to the knackers, and two or three other bullocks were found to be showing symptoms of pain, very dull, grinding teeth, and tail tucked between the legs, and profuse diarrhœa. These animals all recovered.

I went to see the owner of the animals, who lived at another farm, and informed him that no doubt the bullocks were suffering from some form of poisoning, and as it was already dark when I saw these animals I said I would at once make a post-mortem on the dead bullocks, and next day examine everywhere where the bullocks had been, and try and find out the cause of the trouble. Whilst chatting with the owner I casually mentioned what I have just recorded, when he remarked it was an extraordinary thing you should have mentioned

these cases, for there is an old disused mill standing on one side of the yard where the bullocks are, and the same case may apply here.

On a post-mortem I verified his statements, as I found ample evidence of the cause of the trouble in the form of small pieces of white cloth mixed up in the ingesta of the stomach ; the stomach and bowels were very much inflamed, due to the lead present. In this case also a gale had been blowing a day or two previously, which had blown pieces of mill sailcloth into the yard where the bullocks were.

I may say it was dark when I arrived to see these animals, and therefore did not note the presence of the old mill.

YEW TREE POISONING.

By HUGH BEGG, F.R.C.V.S.,
County Veterinary Inspector of Lanarkshire.

About three years ago I was called to investigate the cause of death of four two-year-old heifers at grass. On entering the field, I saw that the animals on the field consisted of twelve yearlings in addition to the four dead two-year-olds. The animals had been put on to the field only two days before my visit. Towards one end of the field there was a clump of trees surrounded by an iron fence. Three of the trees overhanging the fence were yew trees, the branches of which had proved accessible to the larger two-year-olds, but were too high for the year-olds to get at. They showed evidence of having been nibbled at.

The moral is that all branches of poisonous trees which are or are likely to come within reach of grazing animals should be lopped off.

It is worthy of note, too, that when animals have eaten only a small quantity of yew, the symptoms of poisoning do not appear to any marked extent until the act of rumination has been performed.

RHODODENDRON POISONING IN CATTLE.

By Wm. LOTHIAN, M.R.C.V.S.,
Duns.

On March 7, 1917, I was called to see a lot of cattle, over thirty in number, which were in a park alongside which was the avenue leading to a manor house. A gate into the avenue from the field had been left open the day before, and all the cattle got through and had eaten the rhododendron leaves, and were found ill in the morning. I may say the rhododendron were plentiful. Symptoms were dullness, animals looked sick, slavering from mouth and nose, crossed the hind legs when walking, but none paralysed or showed

brain symptoms, slight diarrhœa, and vomiting. What struck me most when going amongst them was the quantities of half-masticated grass, hay and rhododendron leaves which had been ejected from the stomach. They were over the vomiting before I saw them. We decided to leave them alone, as any doubtful benefit from physicing might be lost or counterbalanced from disturbing them. Next day they were very much better. They all made a good recovery, and were none the worse of their experience, which is very different from yew poisoning.

CASTOR OIL SEED POISONING IN CATTLE.

By PHILIP VINCENT, M.R.C.V.S.,
Romford.

Some years ago, late in the evening, I was sent for to go to see some cows, which my client said were dangerously ill. On my arrival I found seven cows which were all purging and straining violently, each animal had an anxious and depressed expression, pulse weak, and increased infrequency, with considerable abdominal pain and intestinal rumblings, no appetite, rumination suspended. I diagnosed poisoning. I gave each cow a dose of Chlorodyne and 4 ounces whiskey and cold flour gruel, and left another dose of Chlorodyne to be given later on. The next morning I found my patients a little better, continued Chlorodyne, whiskey and gruel, and in a few days my patients were convalescent. My client was in the habit of buying the sweepings from the sheds and warehouses in Tilbury Docks, and when I examined his most recent purchase, I found mixed with the other grain a large number of castor oil seeds. These seeds were undoubtedly the cause of all this trouble.

BRACKEN POISONING IN PIGS.

By JAMES BLAKEWAY, M.R.C.V.S.,
Stourbridge.

I have lately had a good deal of trouble with pigs kept in the open on sandy soil where there is a lot of bracken. In all the cases the pigs were in good condition, but the stomach and bowels were more or less inflamed and contained a lot of bracken and sand. They have been kept on the same place for some months, and till the bracken began to get dry we have had no trouble. Is the bracken or the sand the cause? In over forty years' practice I have seen many cases of what we called bracken poisoning, and in most of these there was soon an end to the trouble, when the cattle were put where they could not get bracken. As I write I call to mind a bunch of yearlings and two-year-olds being put on a hill where there was a lot of dry

bracken and very little grass at the end of a very dry summer. In a few days two were dead. I made a post-mortem of these two and found the stomachs and bowels contained a lot of bracken, with numerous hæmorrhages in the abomasum and bowels. The others were at once moved and a mixed purgative given, followed for some days with plenty of linseed tea, with the seeds strained off, and we had no more deaths.

I don't ever remember trouble from green bracken. Of course, I know we are not clear as to the exact cause of the trouble, but that does not prove there is no such thing as bracken poisoning.

TOXICOLOGY NOTES.

By G. MAYALL, M.R.C.V.S.,
Bolton.

What a mine of information veterinary surgeons could produce if all accounts of the cases of poisoning in animals that they encounter during their lifetime were faithfully recorded. The annals of poisoning in human beings would pale into insignificance before the vast array our profession could produce. The Lucretia Borgias, the Palmers, and the Neil Creams flitting spasmodically and luridly on the human horizon would be extinguished and swamped by the steady and regular flow of toxicological deaths on the animal horizon. The harmful drugs of a poisonous nature given in a haphazard and ignorant way by the laity cause no end of deaths. The fetish of a drug (and one often as old as the hills) causes more harm than the parrot cries of politics repeated down the long ages without present and up-to-date thought being exercised upon them. If we had to state the most culpable and ancient drugs given wrongly, by rule of thumb, and in over doses by the laity, we should name syrup of buckthorn, asafetida, turpentine, santonin, tobacco, and castor oil. There are many others that bring us patients occasionally, but the old saw about "trying it on the dog" is still very prevalent to-day.

Strychnine and Digitalis Poisoning.

An Airedale dog with difficulty in swallowing, and pulse 120, respirations 35, temperature 104°, was given some fever medicine consisting of pot. chlor., pot. mt., and liq. am. acet. Two days after it showed improvement, and was given a solution of acetate of ammonia and glycerine. Three days after it was put on medicinal doses of Parrish's chemical food. As it did not eat well, and seemed in a languished state, and the heart beats being weak, the following medicine was prescribed: liquor strychninæ 1 drachm, tincture digitalis 5 drachms, glycerine ½ ounce, water to 2 ounces. A teaspoonful three times daily. It was dead next day.

Phosphorus Poisoning in the Fowl.

1. A Rhode Island Red fowl sent for post-mortem. On opening the crop the strong characteristic smell of phosphorus was present, and the mucous lining was inflamed; section of the gizzard gave the same but fainter smell of phosphorus, and the kidneys were acutely inflamed. We generally get two or three cases of phosphorus poisoning sent to us for post-mortem during the rat campaign week, and there is no doubt that phosphorus ought to be placed out of reach of the domesticated animals.

2. A white Leghorn fowl. On post-mortem the smell of phosphorus was more present on section of the gizzard than on that of the crop. The liver showed fatty degeneration, and the kidneys were inflamed. In this case the owner dropped half a boxful of matches into the drinking water of the fowls when going to the hencote at night. He did not remove them, and the fowls drank of the water, and one or two of them died as a result.

Lead Poisoning in a Cat.

Was called to attend a favourite cat "Prinnie." She had been suffering from vomiting and diarrhœa, and at the time I saw her was convulsed and comatose. She died after a three days' illness. We made a post-mortem, which showed the stomach to be empty, the bowels inflamed throughout their length, and filled with a pasty mass. The owner wished for an analysis of the contents of the viscera, so a portion of bowel, cæcum and stomach were sent to Professor G. W. Clough, D.Sc., Royal Veterinary College, Camden Town. He reported that "definite traces of lead were found to be present, but the amount separated was not more than $\frac{1}{200}$th grain per ounce of material. Death was probably due to lead poisoning."

There had been a lot of painting going on inside and outside the house, and some heavy rain, and probably the cat had drunk a solution of white lead, paint, or putty.

Reviews

The Topographical Anatomy of the Limbs of the Horse. By O. Charnock Bradley, M.D., D.Sc., M.R.C.V.S., Principal Royal (Dick) Veterinary College, Edinburgh. Lecturer on Comparative Anatomy University of Edinburgh. W. Green and Sons, Limited, Edinburgh. 1920. Price, 21s.

This work forms one of the Edinburgh Veterinary Series—some of which have already appeared and others are still in preparation.

Although primarily a dissection guide for students, the book should appeal to all veterinary surgeons. It starts, as it were, *de novo*, for the nomenclature is modern, and no reference is made to any of the misleading

synonyms of years ago. Veterinary anatomy is thus brought into line with other branches of the science, and perhaps in time the doubtful compliment of referring to terms used by us as "of veterinary anatomists" will now disappear. Many will feel the loss of names such as *perforatus* and *perforans*. Our nomenclature in the past has been full of slang. Dr. Bradley has eliminated practically all of it, but has been compelled to retain a few terms much against his will. These, such as chestnut, ergot, foot, frog, bars, toe, quarters, heels, and stifle joint, have been placed in inverted commas, in order to hint that had the author's own inclination had sway, they too would have gone. Horse-owners, however, have popular names for many structures, and these the veterinary student has to learn at some time or other. The profuseness of illustrations becomes apparent when it is stated that there are 115 spread over 172 pages. They are models of accuracy and artistic skill. A feature of great utilitarian value lies in the fact that twelve transverse sections of each limb, made at fairly regular distances, are fully illustrated. The rising generation of English-speaking veterinary students owe the author and his publishers a great debt for this work.

A. W. N. P.

Médecine et Chirurgie Canines. Par P. J. CADIOT, Directeur de l'École d'Alfort, and F. BRETON, Ancien chef de travaux de chirurgie à l'École d'Alfort, Vétérinaire à Paris. Quatrième Édition. Avec 72 figures dans le texte. Paris: Asselin et Houzean. Price 20 francs.

THE fourth edition of this little book of 420 pages, with 72 figures in the text, forms one of the best and cheapest clinical guides on Canine Medicine and Surgery in any language. It is written in a concise and lucid style, so that any English-speaking veterinary surgeon with only a little knowledge of the French language should be able to follow the text with profit to himself. The illustrations are very graphic of the conditions they are intended to depict. Prescriptions are freely interspaced throughout the text, which makes the work particularly valuable for clinicians. The section on Tuberculosis should especially appeal to the up-to-date practitioner, as although the disease is far from rare in the dog it is seldom recognised, at least in most of its phases or forms.

There is no useless padding, and what one observes is the fact that personal pronouns, singular or plural, are conspicuous by their absence.

The authors deserve well of their profession, and one of them, Professor Cadiot, has a world-wide reputation, which is a sufficient guarantee of the quality of the material embodied in the pages of the volume. Therefore we have no hesitation in recommending it to our English-speaking confrères, who nowadays have to depend so much on the treatment of the diseases of the dog as a source, and, in many instances, the chief source, of income.

NOTICES

All communications should be addressed to 8 Henrietta Street, Covent Garden, London, W.C. 2. Telephone: Gerrard 4646. Telegrams: "Baillière, Rand, London."

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